The Significance of an Absent Radial Pulse in Supracondylar Fracture of the Humerus

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THE correct method of treatment of a supracondylar fracture of the humerus with no palpable radial pulse still presents a problem. There is obviously still some doubt as to whether the absent radial pulse should give rise to any alarm, provided the hand is a reasonable colour, and the circulation in it seems to be quite satisfactory. The author feels that it should, and it is the purpose of this paper to encourage others to undertake early exploration and exposure of the brachial artery at the fracture site in these cases.

Griffiths (1940) has already made it quite clear that in Volkmann's ischæmic contracture the basic pathology is a necrosis of muscle due to an insufficient supply of arterial blood. This necrosis is an irreversible change, and occurs when the blood supply has been absent for six to eight hours. After that time nothing can prevent the necrotic muscle from being replaced by fibrous tissue and an ischæmic contracture developing. It is therefore clear that if there is a likelihood of such necrosis occurring, and I believe this to be so in all cases where there is no palpable radial pulse, then something must be done within eight hours of the injury. The first step is, of course, to reduce the fracture, as this alone may restore an absent pulse. If this fails, the arm should be extended, if necessary until it is quite straight. If there is still no pulse, then, no matter how good the circulation in the hand appears, the brachial artery should be explored.

Three cases of supracondylar fracture with no radial pulse have been seen in the last two years.

> **Case 1.**—A 7-year-old boy was admitted to hospital six hours after sustaining a supracondylar fracture of the right humerus with backward displacement of the lower fragment. On admission, there was no radial pulse and the hand was a little dusky but quite warm. The fracture was satisfactorily reduced about eight hours after injury, the arm being held in full flexion as is necessary to maintain reduction. The pulse did not return, and so the arm was slowly lowered until it was fully extended, but the pulse remained absent. The circulation in the hand was reasonably good, however, and it was decided to leave the arm fully extended in a light P.O.P. back slab in order not in any way to impede the collateral circulation. When I first saw him some forty hours after the injury, a weak pulse was palpable and the circulation in the hand was fair. The collaterals were now functioning well. Already, however, the fingers could not be fully extended with the wrist dorsi-flexed, and he went on to develop a full and severe Volkmann's ischæmic contracture.

In this case the blood supply during that crucial eight hours had been insufficient to prevent necrosis of the muscle fibres occurring. When the pulse is absent, but the hand circulation appears adequate, it is clear that the brachial artery itself is

no longer patent, but that the collaterals are supplying enough arterial blood to satisfy the skin of the hand. The essential point, however, is that the muscles of the forearm should be adequately supplied with arterial blood within six to eight hours of the injury, and this does not seem to be the case if no radial pulse is palpable.

Case 2.—The patient, a girl aged 6, was admitted and seen by me four hours after having sustained a supracondylar fracture of the left humerus, with marked backward displacement of the lower fragment (Figs. 1 and 2). The radial pulse was absent, the left hand was cooler than the right and a little paler, but there was a reasonable capillary return. A good reduction was obtained under a general anæsthetic, by strong traction on the extended arm, followed by flexion of the elbow with forward pressure on the lower fragment. The pulse, however, was still absent. The arm was therefore slowly extended, but the pulse did not return. At this stage 4 c.c. of 1 per cent. procaine was given intravenously without effect. Exposure of the brachial artery was then undertaken.

The skin incision was in all about six inches long, being longitudinal above and and below and transverse at the skin crease of the anticubital fossa. No veins were encountered, and there was practically no bleeding. The deep and bicipital fascia were divided, and some blood clot removed. This was small in amount, and could not have been occluding the artery by pressure. The tendon of the biceps was identified, and medial to this what was taken to be the median nerve was clearly seen. The brachial artery should lie between these two structures, but a careful search failed to reveal it. It was then thought that possibly it was not the median nerve that had been found, but the artery in spasm. Fully two inches of it could be seen proximal to the fracture, and it all had the appearance of nerve, and there were no pulsations. However, it was decided to clear it fully and dissect it free, and almost as soon as this was begun, it dilated markedly and began to pulsate. It was soon pulsating strongly, but the anæsthetist was unable to report any pulse at the wrist. It was then seen that the pulsations ceased where the artery ran over the jagged lower edge of the upper fragment, and it soon became apparent that the artery, or its adventitia, was actually impaled on this jagged bone edge. It could not be freed by blunt dissection, and there was a tight band round the artery at this point, exactly as if it had been tied with a piece of thread. Presumably this was a band of adventitia pulled tight by being firmly impaled on the bone edge behind. It was only when a knife was used to cut between the bone and the artery that the artery could be freed. The tight ring was now no longer present and the whole artery was pulsating, with a palpable pulse at the wrist. The arterial wall itself, on inspection, appeared quite uninjured, although it is hard to see how it escaped being nipped during reduction of the fracture. The wound was then closed, skin only being sutured, and the arm kept extended in a back slab. The operation ended exactly eight hours after the injury had been sustained.

An X-ray on the following day showed that the reduction had not been maintained. This is only to be expected, as the arm is necessarily extended during and after the operation. Reduction was at once attempted, but was not successful, as one was not prepared to use much force for fear of damaging the all-important brachial artery, and also, every time the arm was flexed, the pulse vanished completely. Accordingly, the arm was left extended with marked backward displacement of the lower fragment, but with a good pulse.

This position was maintained for one week, by which time much of the swelling had subsided, and it was judged safe to make a proper attempt at reduction. Closed reduction was attempted, but the fragments could not be moved due to early callus formation. A small longitudinal incision was made posteriorly down to bone over the lower end of the humerus, and using a bone lever like a shoe horn, the lower fragment was levered forward into good position. (This is not a difficult procedure, and has subsequently been used on other cases where closed reduction had failed.) The pulse,

however, still became very weak if a position of extreme flexion was adopted, and so the arm was put up in a sling with the elbow flexed to a right angle only. Even at this angle the pulse was appreciably weaker than the good side, but in half an hour it had completely recovered. An X-ray showed good position. Two days later a further X-ray showed that the deformity had recurred, proving that such a fracture is only stable in extreme flexion. Closed reduction was at once attempted, and was successful, as the fragments could now be easily moved. A position of full flexion was maintained this time with elastoplast and a collar and cuff sling. The pulse was at first very weak, but in two hours was back to normal. There were no further mishaps, and union occurred in good position. As can be seen from the photographs taken about a year later, a normal arm and hand was the end result (Figs. 3, 4, 5, and 6).

Case 3.—A boy, aged 5, was admitted to hospital suffering from a supracondylar fracture of the humerus, with backward displacement of the lower fragment. The fracture was immediately reduced, a very satisfactory position being obtained. The hand was noted to be warm, a little dusky, but with a good capillary return. There was no palpable radial pulse either before or after reduction. I first saw the child about twenty hours later, and even at that time ischæmic contracture of the flexor muscles could be easily demonstrated, and it was clear that the irreversible necrosis of the muscle fibres had already taken place. The child has since developed a Volkmann's ischæmic contracture.

Discussion.

It is the aim of this paper to show that in cases of supracondylar fracture of the humerus it is the state of the radial pulse and that alone which indicates whether or not a Volkmann's contracture is likely to develop later. In Cases 1 and 3 the fact that there was no radial pulse was fully appreciated at the first examination, and the possibility of a subsequent contracture developing was considered. In both cases, however, the circulation in the hand and fingers was such that the surgeons felt confident that there must be an adequate circulation to satisfy the needs of the forearm muscles. In both cases this assumption was proved wrong, and one must assume therefore that the state of the circulation in the skin of the hand is no guide to the state of the circulation in the forearm muscles. The only guide to this is the state of the radial pulse, and it seems clear that where this is completely absent, muscle circulation will be inadequate. No conclusion can be drawn from these cases as to what should be done if the pulse is present but appreciably weaker than the good side. Griffiths gives as his indication for exploring the artery an absent or a very weak pulse, but also states that the presence of the radial pulse at all in the immediate post reduction period is a definite assurance that a severe Volkmann will not occur. One feels therefore that a weak pulse should only be taken as an indication for operation if it is so weak that there is, in fact, some doubt as to its presence at all.

Another important aspect of the prevention of contracture is the urgency with which operation on the artery must be undertaken. The irreversible change in the muscles is thought to occur about six to eight hours after the blood supply has been cut off. In the second case the blood supply was restored eight hours after the injury, and there were no ill effects. In Case 3, however, actual contracture was beginning twenty hours after the injury, and it is clearly useless at this stage to try and improve the blood supply. Somewhere between eight and twenty hours

after the injury the "point of no return" is reached. Exactly when it is reached is not known, and will certainly vary from case to case. Griffiths had success up to seven hours after injury, and failure at twenty-three hours. We have had success at eight hours, and failure at twenty. Undoubtedly, therefore, this is an operation which must be performed at the earliest possible moment, and certainly within the first eight hours. As to the value of operation after this time one is only guessing, but provided there is no contracture already present, it would seem to be worth while exploring the artery up to say twelve or fifteen hours after the injury.

Finally, there are some definite facts which can be learned from a consideration of the findings in the second case.

First. That apart from laceration or complete tearing of the brachial artery, about which little can be done, there are at least three causes for an absent radial pulse after a supracondylar fracture, all of which can be overcome and relieved.

(a) The maintenance of too much flexion at the elbow joint in the presence of swelling. This is especially dangerous when the reduction has been carried out very soon after the injury and before the swelling is maximal. While this is the ideal time to do the reduction, further swelling will occur and may obliterate later a pulse that was normal immediately after reduction. If, on the other hand, reduction is being carried out some days after the injury, as it was in this case, it was found that while extreme flexion might greatly weaken the pulse for a time, it always recovered in about an hour.

(b) The artery may be mechanically obstructed by the sharp lower edge of the upper fragment.

(c) The artery may be in spasm.

Second. That a supracondylar fracture is unstable and the deformity will recur unless the elbow is in a position of full and extreme flexion. Plaster of paris back slabs seem to be of little value if the elbow is only flexed to a right angle, whereas if the elbow is flexed to its fullest extent, the triceps tendon holds the fracture stable, and no plaster is needed.

Summary.

Three cases are described illustrating the danger of Volkmann's contracture developing after a supracondylar fracture of the humerus in all cases where the radial pulse is not palpable, even though the circulation in the hand appears good. Operative exposure of the artery has been successful in preventing this, the operation being performed within eight hours of injury. Developing contracture has been seen within twenty hours of injury.

I wish to thank Mr. H. P. Hall, M.Ch., for his permission to publish these cases, and for his advice and support in the treatment of the second one.

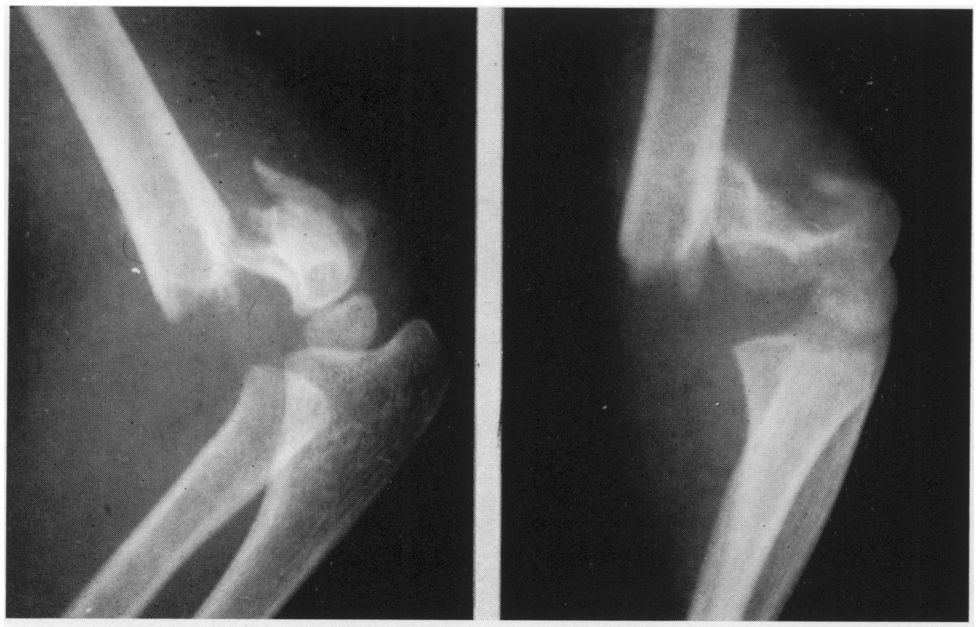

Fig. 1. Fig. 2.

Lateral and A.P. views to show the original deformity in Case 2.

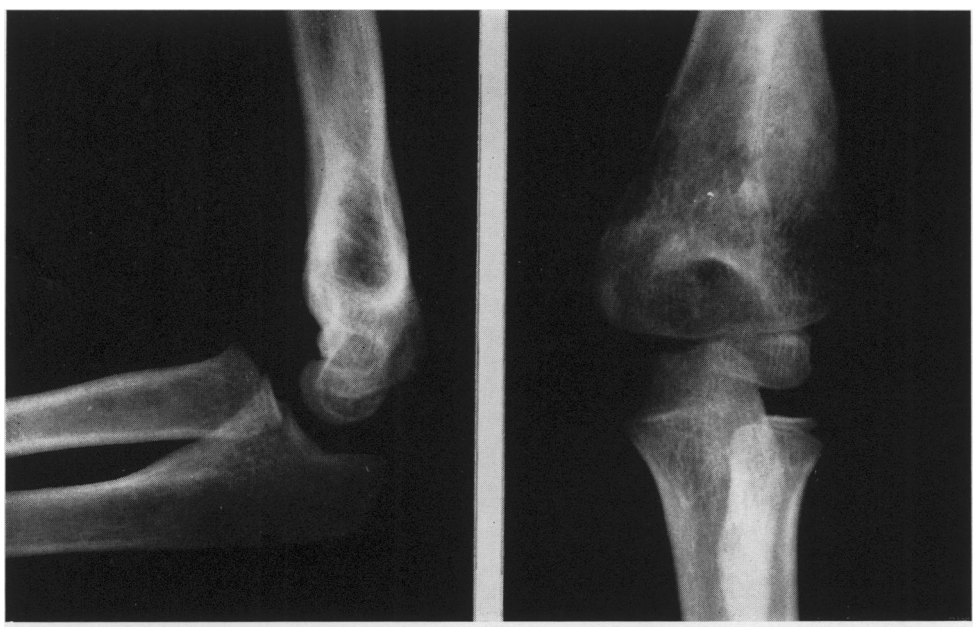

Fig. 3. Fig. 4.

Lateral and A.P. views to show the final result in Case 2.

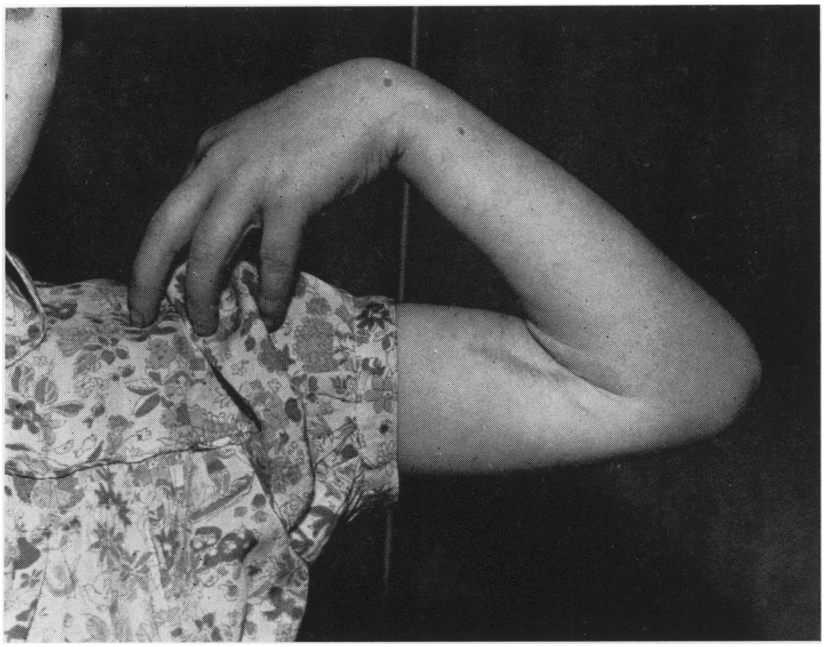

Fig. 5.

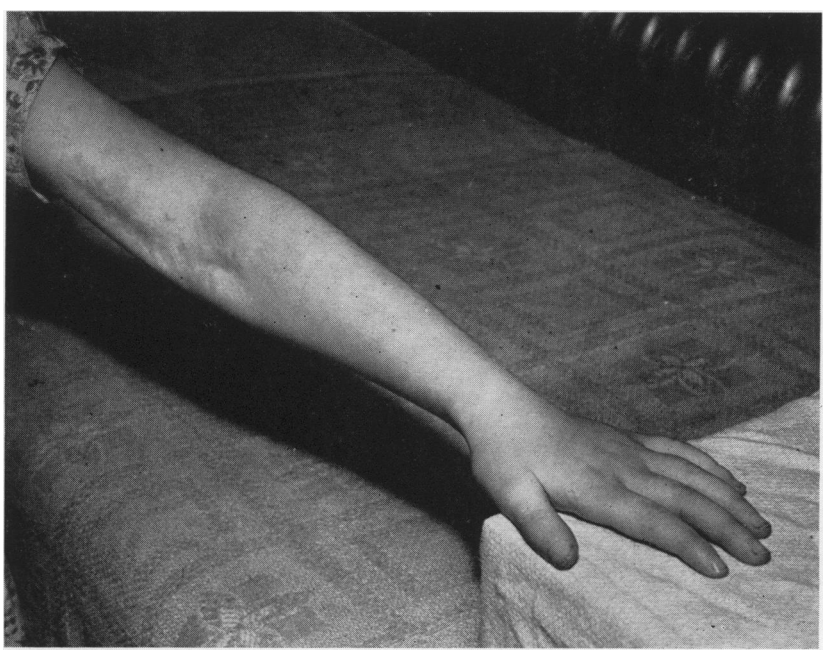

Fig. 6.

These pictures show the normal range of movement at the elbow joint, one year after the injury. Notice also the normal hand, and the incision used to expose the brachial artery.